published in your Journal [1]. The authors suggest that 27% of the eating disorders patients meet criteria for seasonal affective disorder (SAD).

Serotonergic system plays an important part in feeding regulation [2] and several lines of evidence suggest that serotonergic system is involved in the pathogenesis of SAD [3]. Thus, researchers focus on determining whether variants of genes related to serotonergic transmission are associated with SAD and eating disorders.

We have recently reported an association between the −1438G/A promoter polymorphism of the 5-HT2A gene and SAD [4]. We found a significant increase in the frequency of the −1438A variant allele of the 5-HT_{2A} promoter polymorphism in SAD patients compared to low-seasonal controls. The difference in genotype distribution was also significant.

Many, but not all studies have found an association between the −1438G/A 5-HT2A polymorphism and eating disorders. This association has been reported by Collier et al. [5], Sorbi et al. [6], Enoch et al. [7], Nachmias et al. [8] but not by Hinney et al. [9], Campbell et al. [10], Ziegler et al. [11]. Despite the discrepant results of the studies it is very possible that the −1438G/A promoter polymorphism of the 5-HT2A gene plays a role in the development of eating disorders.

Levitan et al. [12] have recently reported preliminary results of their ongoing study. The authors suggest that variation of the tryptophan hydroxylase gene may play a role in eating behavior and weight regulation in females with SAD.

Thus, it is possible that there is a genetic link between SAD and eating disorders. It is interesting to speculate that there are specific inherited personality types with a predisposition to both eating disorders and SAD. Obsessional, perfectionistic, and anxious personality traits may contribute to the pathogenesis of eating disorders and SAD.

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Measuring care needs and case-mix by means of the INTERMED

To the editor:

In a recent issue of this Journal, Dr. Katon described the importance of conducting treatment trials in real world settings as opposed to efficacy studies focused on providing intensive treatments to carefully selected patient populations [1]. The necessity of this shift to effectiveness studies lies in the limited access to optimal treatment—less than half of the patients with hypertension, diabetes or depression receive adequate treatment—and the increasing percentage of complex patients suffering from multiple chronic diseases and/or psychiatric comorbidity. Ideally, in a real world setting, a clinician would obtain information about a patient's biopsychosocial disturbances and the ways they might interact with current and long term treatment in order to formulate an adequate strategy. Likewise, in effectiveness studies, a clinical researcher would obtain this information in order to make an appropriate estimation of treatment effect, or to select patients for whom additions to standard treatment should be formulated.

In recent years, we have developed an instrument, called the INTERMED, which can be utilized as a case-mix instrument for clinical and research purposes [2,3]. The INTERMED enables a biopsychosocial assessment of the patient's past, current and future care needs by scoring 20 risk factors on a manual-based patient interview of about 15 minutes. First studies demonstrated a satisfying inter-rater reliability and validity with regard to relevant parameters in different patient populations with varying somatic and psychosocial care needs.

Studies of patients with chronic low back pain and diabetes suggested that patients identified by the INTERMED as being biopsychosocially complex responded not as well, by far, to standard biomedical treatment than the less complex patients. We will now evaluate in prospective intervention studies whether these complex patients will benefit from multi-modal treatments, consisting of somatic, psychiatric and social care.

The delivery of health care in real world settings, as Katon mentioned, should be focused on individual care needs. For this goal, the INTERMED can be used as a clinical decision-making tool. In order to assess the effectiveness of treatments in real world settings, the INTERMED can be used to control for confounding variables and designing multimodal treatment.

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Dermatologic presentation of panic disorder

To the Editor:

Several investigators have suggested a classification system for panic disorder based upon predominant symptom clusters. In this regard, Bouwer and Stein [1] classified patients according to cardiovascular, respiratory, gastrointestinal, and occulovestibular symptoms. Other investigators have described depersonalization-derealization [2], derealization [3], and cognitive [4] subtypes of panic. Whether these symptom-based subtypes demonstrate epidemiologic, etiologic, or treatment differences remains unknown. However, among some individuals, panic disorder appears to present as specific and predominant clusters of somatic and psychological symptoms. We wish to report a case of panic disorder that was highlighted by dermatologic symptoms.

Case report

A 19-year old, white male presented to the outpatient internal medicine clinic for evaluation of an intermittent rash which had been occurring over the past 6 weeks, up to 2 to 3 times per day, and lasting 10 to 15 minutes. The rash initially emerged on the patient's chest and back, and promptly extended to all extremities. The rash was pruritic, maculopapular (1–2 mm), erythematous, nonvesicular, and non-urticarial, and described as "pins and needles" in sensation. Adjunctive symptoms included tachycardia, feelings of "disorientation," inability to focus, and restlessness. The patient denied dyspnea, nausea, dizziness, diaphoresis, perceptual disturbances, diarrhea, or headaches. The symptoms began after the termination of a significant relationship and were subsequently triggered by various emotional precipitants. With each outbreak, the patient would "talk himself down," physically relocate himself, shower, chew gum, or place his hands in cold water. He expressed his social concern about the visibility of the rash, stating that coworkers had commented upon his dramatic symptoms.

During initial consultation with his primary care physician, the patient developed the rash while waiting in the examination room. Vital signs including blood pressure were normal. Laboratory studies indicated no eosinophilia or thyroid abnormalities. The patient was started on cetirizine and paroxetine beginning at 5 mg per day.

During psychiatric consultation 10 days later, the patient reported no prior mental health exposure, but acknowledged a history of low-grade anxiety and a "panic attack" in the 6th grade which was not accompanied by dermatologic symptoms. He described himself as a "worrier" by nature and had been placed on fluoxetine during mid-adolescence by a primary care physician for the treatment of anxiety. The family history included substance abuse, antidepressant exposure, and one suicide, but no panic disorder. The dose of paroxetine was gradually titrated to 30 mg per day and at a follow-up visit 4 weeks into treatment, the patient experienced complete remission of all symptoms.

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